EXPERIMENT 33, MARCH 30, 1906.

T.'s white blood corpuscles... 1 vol.
J. P.'s serum........................... 1 vol.
Suspension of tubercle bacilli 1 vol.
(The whole incubated at 37° C. for twenty minutes.)

1st	25	polynuclear cells	= 113 t. b.	= average of 4.52 t b. per cell
2d	"	" "	" 97 "	" 3.88 " "
3d	"	" "	" 88 "	" 3.52 " "
4th	"	" "	" 109 "	" 4.36 " "
5th	"	" "	" 98 "	" 3.92 " "
6th	"	" "	" 115 "	" 4 60 " "
7th	"	" "	" 115 "	" 4.60 " "
8th	"	" "	" 114 "	" 4.56 " "
1st	50	polynuclear cells	= 210 t. b.	= average of 4.2 t. b. per cell
2d	"	" "	" 197 "	" 3 94 " "
3d	"	" "	" 213 "	" 4.26 " "
4th	"	" "	" 229 "	" 4.58 " "
1st	100	polynuclear cells	= 407 t. b.	= average of 4.07 t. b. per cell
2d	"	" "	" 442 "	" 4.42 " "
1st	200	polynuclear cells	= 849 t. b.	= average of 4.24 t. b. per cell

EXPERIMENT 33, MARCH 30, 1906.

T.'s white blood corpuscles... 1 vol.
K.'s serum............................ 1 vol.
Suspension of tubercle bacilli 1 vol.
(The whole incubated 37° C. for twenty minutes.)

1st	25	polynuclear cells	= 100 t. b.	= average of 4.00 t. b. per cell
2d	"	" "	" 103 "	" 4.12 " "
3d	"	" "	" 58 "	" 2.32 " "
4th	"	" "	" 77 "	" 3.08 " "
5th	"	" "	" 115 "	" 4.60 " "
6th	"	" "	" 81 "	" 3.24 " "
7th	"	" "	" 84 "	" 3.36 " "
8th	"	" "	" 96 "	" 3.84 " "
1st	50	polynuclear cells	= 203 t. b.	= average of 4.06 " "
2d	"	" "	" 135 "	" 2.70 " "
3d	"	" "	" 196 "	" 3.92 " "
4th	"	" "	" 180 "	" 3.60 " "
1st	100	polynuclear cells	= 338 t. b.	= average of 3.38 t. b. per cell
2d	"	" "	" 376 "	" 3.76 " "
1st	200	polynuclear cells	= 714 t. b.	= average of 3.57 t. b. per cell

A SUGGESTION IN THE TREATMENT OF HÆMOPTYSIS.[1]

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THE cause and treatment of pulmonary hemorrhage have interested physicians since the time "when the memory of man runneth not to the contrary." Its importance in pulmonary tuberculosis has long been known, though it is more recently that its true relation to the disease has been established. Long a moot point in medicine, hæmoptysis has been held by many famous authorities, among them Hippocrates, Galen, Aretæus, Hoffman, Boerhaave, Van Swieten, Graves, and Niemeyer, as a precursor and even the cause of pulmonary tuberculosis. Opposed to them stood Richard Morton, Portal, Bayle, Laënnec, and Louis. Walshe, in the middle of the last century, showed clearly that Laënnec and Louis were correct in holding that in most instances symptoms preceded the hæmoptysis which should, therefore, be looked upon rather as the result than as the cause of the pulmonary tuberculosis. A few years later, 1868, Rasmussen, of Copenhagen, published in the *Edinburgh Medical Journal* his pathological researches upon the origin of hæmoptysis. He showed, as is well known, that fatal hæmoptysis proceeds from a ruptured aneurysm in a pulmonary vessel. Since then it has been clearly established that fatal hæmoptysis is due in practically all cases to the rupture of an aneurysm situated upon a branch of the pulmonary artery. Few, I might say almost no, well authenticated cases of fatal hæmoptysis have occurred from rupture of the pulmonary vein or of the bronchial artery. It has long been known that the walls of the pulmonary vessels are quickly attacked in tuberculosis. The endarteritis itself is usually obliterating. Even when this does not occur the vessel walls are thickened and immobile.

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[1] Read at the meeting of the National Association for the Study and Prevention of Tuberculosis, Washington, D. C., May 17 and 18, 1906.

The pulmonary blood pressure may be affected by change in the calibre of the bloodvessels, in the greater or lesser circulation, by increased or lessened flow of blood, by change in the rate of circulation, and by failure of the left side of the heart to perform properly its function. Recent work has thrown much doubt upon the existence of vasomotor nerves in the pulmonary vessels and it is probable that, cæteris paribus, the pulmonary pressure is directly determined by the amount of blood supplied to the right ventricle.

Theoretically, hæmoptysis may be controlled by increasing the coagulability of the blood, by lessening the rapidity of its flow, by reducing the volume of blood in the weakened vessel, by lowering the blood pressure, or by constricting the affected vessel, which may act in one of the ways heretofore mentioned. Calcium chloride has not accomplished in my hands what I was led to hope for from Wright's experiments. Digitalis has often signally failed to be of any benefit. The pathological changes occurring in the pulmonary vessels in the diseased area preclude any changes in their calibre. All drugs, therefore, which cause a contraction of the bloodvessels, such as ergot or adrenalin, would, if they acted upon the pulmonary vessels, be directly injurious, as they would cause a contraction of the healthy vessels, and the diseased vessels, incapable of varying in diameter, would be subjected to increased instead of lessened tension. Recent experimental work, which has been mentioned, would seem to indicate that lessened tension from dilatation of the other pulmonary vessels does not take place.

For these reasons we must seek elsewhere to regulate the pulmonary tension, and in considering the subject the first thought is to lower the tension by reducing the volume of blood in the circulation. All severe hæmoptyses act quickly in this way and in most instances render venesection useless and even dangerous. Another attempt in the same direction is ligation of the limbs, a modern modification of Junod's boot.

More recently, however, a group of substances has been brought into use which exert a very marked effect upon the blood pressure. I refer to the nitrites. Their action consists chiefly in producing a dilatation of the peripheral vessels, mainly of the head and neck, and of the vessels of the splanchnic area, causing a marked fall in the blood pressure while the heart is accelerated. The different members of this group vary in regard to the rapidity, the intensity, and the duration of their action. Amyl nitrite acts instantaneously, but only for about five minutes. Nitroglycerin, and in fact all the other nitrites, act less vigorously but much longer. Nitroglycerin acts more quickly and more intensely, but for a slightly shorter period than sodium nitrite. In both the action is well marked in fifteen minutes and lasts for one to two hours. Erythrol tetranitrate acts much more slowly and attains the height of its action in about two hours, but its duration is much longer.

Theoretically, amyl nitrite should be administered at once in the case of hæmoptysis and repeated if necessary in five minutes. Following the first dose nitroglycerin or sodium nitrite should be given, and in a few minutes (twenty to thirty) erythrol tetranitrate should be given. This would insure a lowered pulmonary tension for some time. The great difficulty, however, lies in the fact that it is difficult to tell just when to repeat the dose. It is dangerous to lower the blood pressure too much and dangerous not to lower it enough. To overcome these difficulties I have recently taken the patients' blood pressure[1] every two hours and ordered the doses accordingly, aiming to keep the tension within certain limits. Practically, I use amyl nitrite as suggested, and if the patient is nervous administer morphine (one-eighth grain) hypodermically and along with it nitroglycerin or, which I prefer on account of its greater stability and fewer untoward symptoms, sodium nitrite (one grain). In some cases the patient cannot inhale amyl nitrite, and in these cases the sodium nitrite or nitroglycerin should be given hypodermically, immediately, and repeated as often as necessary. A careful nurse may be quickly trained to take accurate readings of the sphygmomanometer and the cuff may be left upon the arm indefinitely if preferred. To determine at which level to keep the blood pressure I take it several times at short intervals and attempt to keep it usually between "100 and 115 or 120 mm. of mercury." It is comparatively easy to do this with the aid of sodium nitrite.

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[1] The systolic pressure only has been studied.

Hæmoptysis frequently recurs or even occurs in the early morning hours and the patient awakens spitting blood. Howell's theory of sleep offers a striking explanation of this phenomenon. This observer holds that sleep is due to the fatigue of the vasoconstrictor centre (or centres), and he has shown by the plethysmograph that a dilatation of the peripheral vessels occurs during sleep. In the early morning hours, when the vasoconstrictor centre is regaining its lost tone, remarkable vacillations occur in the plethysmographic records, which would indicate considerable variation in the quantity of blood supplied to the right side of the heart and, as we have mentioned before, concomitant variations in the pulmonary blood pressure. Sudden variations in pressure are, in all probability, more dangerous than a steady high pressure. To prevent these variations as much as possible and to equalize the blood pressure, I have recently given morphine and sodium nitrite hypodermically between midnight and 2 A.M., waking the patient if necessary.

Aconite has been suggested in the treatment of hæmoptysis and its use, when controlled in the foregoing manner, may prove to be very beneficial. I would suggest its use in cases complicated with fever.

To recapitulate briefly, I would suggest that the blood pressure be frequently observed, that morphine be used when necessary to quiet the patient and so equalize the blood pressure, that sodium nitrite be exhibited to reduce when necessary the blood pressure, and that in case of a sudden hæmoptysis amyl nitrite be administered at once when possible, to produce a marked fall in the blood pressure and so aid in a temporary cessation, at least, of the hæmoptysis.

In no symptom, or complication, as you wish it, is judgment of the effect of any treatment more difficult than in hæmoptysis. I am fully persuaded that many of our patients recover from hæmoptysis in spite of our treatment. I have had too few cases to hazard anything more than a tentative opinion upon the beneficial effects of this method of treatment. It seems to me, however, a little less empirical than many others and I submit it to you in the hope that any of you who think well enough of it will endeavor to establish its value or to prove its worthlessness.